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A SUCCESSFUL BLOOD BANK IN A NON-TEACHING HOSPITAL*

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Two years ago when the possibility of a blood bank at St. Joseph's Hospital, bed capacity of only 228, was first discussed, its success was quite dubious in the minds of many. Blood banking itself was not universally accepted throughout the country.

We do not have a medical staff with a single head whose policies must be followed, as in university hospitals, but a staff of individuals each acting by his own lights.

Would our turn-over of blood be sufficient? In reviewing the literature about blood banks, it was noted that it is not advisable for an institution to attempt to start a blood bank unless at least three transfusions are given daily[1]; otherwise there would be a great loss of blood due to aging and insufficient number of the various types. Our total number of transfusions for 1938 was 747. That is a questionable sufficiency for adequate turn-over.

In the studies on "stored blood" the question has been raised as

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to its efficacy to combat infection[2], pro-thrombin content[3,4,5]; the disintegration of platelets and white cells[6], toxicity of the diffused potassium from erythrocytes to plasma[7,8]. Each month would find added investigations along these lines with a variety of results.

The idea of giving blood just above freezing temperature was a hard sell when all previous infusions had been warmed to body temperature.

Blood capital had to be secured. From whom? How much stock would be necessary to start? What types? Who would be responsible for the bank's institution, its operation, management, the administrative problems and technical details? These were some of our problems.

Bank

Despite these difficulties, the bank was instituted April 22, 1939. It was made a division of the laboratory and was to operate under the direct management of the laboratory personnel. The superior was most liberal in supplying equipment and did not hesitate to purchase blood when any particular type was low. Support of the staff and cooperation of the interns was invaluable.

When a transfusion is ordered on any patient, he is immediately typed; the family is notified that blood is needed and what approximate number of donors will be necessary. Blood of the correct type previously tested serologically is then taken from the bank, cross-matched to be sure of its compatibility, and if satisfactory, is given to the patient at the time ordered by the doctor. Later, when the donors come in, regardless of what their types or tests may be, they are bled to replace the volume given and to deposit a sufficient amount for future use.

This blood[9] drawn from donors is collected in 1000 cc. pyrex Erlenmeyer flasks containing 50 cc. of 4% sodium citrate as an anticoagulant, and is topped with a two-holed rubber stopper, one hole providing for an air vent, the other an opening for a small piece of glass tubing.

After the required amount of blood has been obtained, the rubber tubing is pinched, the needle withdrawn, the open cork immediately replaced by a solid cork, and the blood remaining in the rubber tubing allowed to drain into two small bottles which are sent to the

laboratory for determination of type and for serological tests. The flask of blood is now properly tagged with name, address, sex, *race,* and brief history as to venereal diseases, allergy, trauma, etc., and placed in a special refrigerator of 4°-6° C., until tests are completed. The satisfactory blood is then transferred to the bank also of 4°-6° C., and placed in its proper division according to types. Unsatisfactory blood is used for blood media, or discarded. Bloods from white and colored are kept separate; hence we might say in reality that we are managing two banks. Public sentiment seems to demand this.

The bank is "housed" in a twelve cubic foot General Electric Refrigerator with double doors. The left side is used for white blood and the right for colored. A device that we use just as a safety measure is to attach blue tags to the flasks of blood collected from colored people and buff tags to those from white.

Blood is not allowed to become too old. When ten days have elapsed since its withdrawal, or if any hemolysis should occur before that time, the plasma is routinely drawn off for plasma infusions.

Each patient, as he is typed, opens an account with the bank. His deposits and withdrawals are recorded on his account-card and in the bank-book ledger. Of every transfusion that is given a statistical record in tabulated form is kept. It includes date, name of patient, type, race, doctor, diagnosis, name of donor, amount of blood given and whether freshly drawn or bank; if bank blood, number of days since drawn, length of time for infusion, why given, reaction, if any, with detailed description.

Benefits

So beneficial did the bank very soon become that expense to patient was cut, blood was available at all times, day or night, for general treatment or emergency, in any amounts; previous prejudices began to be broken down and the bank was utilized more and more.

Charity patients, who in the past were dependent upon the volunteer donor, are given blood as their conditions demand. Since its institution the bank has given 183 charity transfusions without any replacement whatever.

The surgeons gained a feeling of security in being able to call for blood in cases of emergency. Shock is not the dreaded thing it

used to be. At its first sign, plasma is now administered with gratifying results. Lives have been saved, which by the old régime would probably have been lost.

Hospital stay is shortened. The surgical patient is helped over those first post-operative days by transfusions; the septic and anemic cases make a much quicker recovery. This makes for increased efficiency of the hospital which is thus enabled to accommodate more patients per year.

The blood bank saves time, labor, and anxiety for the laboratory; hurried typing, cross-matching, and serological tests are eliminated. The new system is much more satisfactory to all concerned.

To date, after two years of blood-banking, 2724 transfusions have been administered; 2311 to 820 white patients, and 413 to 175 colored patients. Contrast this with the number given in other institutions for a similar length of time:

Hospital	Bed Capacity	Transfusions	
St. Joseph's Hospital Lexington, Ky.	228	1239	(12 mos.)
Iowa State Hospital	900	1604	(15 mos.)[10]
John Gaston Hospital and University of Tennessee	550	1415	(12 mos.)[11]
Gallinger Municipal Hospital Washington, D. C.	1220	2000	(12 mos.)[1]
Jewish Hospital Brooklyn, N. Y.	547	1000†	(annually)[12]

It is of interest to note at the end of the two years that the percentage of reactions of fresh blood and bank blood has been about the same: Bank 3.7%; Fresh 3.2%. The purposes for which the transfusions were given was quite interesting in that 32.5% were for anemia; 24% for sepsis; 18.8% for post-operative treatment; 9.4% for hemorrhage; and, 15.3% for miscellaneous.

The bank is becoming more and more popular, not only in our hospital, but in the surrounding vicinity. It has served five neighboring hospitals on different occasions and, as the months go by, there is a noticeable increase in its utility.

Summary

1. Handicaps in starting the blood bank at Saint Joseph's Hospital are itemized.
2. Method adopted is given.
3. Benefits derived are enumerated.
4. Statistical report for the past two years is presented.

Conclusion

The one secret of our success, we feel, lies in the centralization of authority with power delegated by the staff, yet divorced from it, and responsible only to the superintendent.

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REMARKS ON THE MICROSCOPIC STUDY OF THE BLOOD*

By EMIL SCHWARZ, M.D.

Techniques are but expansions of the capacities of our senses and of our limbs to increase our control of the forces of nature. They can accomplish the desired effect only when handled with understanding and accuracy. The technician's task is to master them. In order to do so he or she has to possess skill, knowledge, intelligence and responsibility. Knowledge is acquired by study and practice; skill, by continued exercise. Intelligence is an inborn gift to be developed by constant use. Responsibility has its fundament in the individual character and is strengthened by the realization of the importance of one's work. Without this intellectual and moral base the work of the technician soon degenerates into a mechanical and stereotyped business. It errs and stumbles over the simplest deviation from the regular pattern. Mere mechanical activity carries with it the danger of a loss of interest in the work, which eventually must become a burden instead of a loved occupation. Any work done without love is inferior.

When I was a young physician (which was many years ago), physicians did all laboratory diagnostic procedures themselves. Due to the progress of science, the quantity and variety of the accessory but nevertheless indispensible means for diagnosis and treatment increased so much, that it is no longer possible for a practicing physician to master the techniques and to spend the time necessary for all these investigations. He must rely on the help of the technician, who has acquired the necessary skill and accuracy and who procures the data needed for his interpretations. The physician must presume that these data are acquired with the greatest care

* *Read at the regular monthly meeting of the Chicago Society of Medical Technologists, December, 1940. Submitted for publication by Cecelia M. Kortuem, M.T. (A. S. C. P.)*

and exactitude. Therefore, the technician shares with the physician the moral responsibility for the patient, a realization of which the technician must be fully aware. This is the source of my high esteem for the profession of Medical Technology and of my appreciation for the invitation to address you at your meeting.

Remarks on the Microscopic Study of the Blood

You do a great deal of your work with the microscope. Just as the telescope extends our sight into immeasurable dimensions of space, so this marvelous instrument reveals the minute world hidden to the naked eye, rendering this part of the work, perhaps the most exciting of your activities.

You see bacteria and other microorganisms; you detect parasites; you learn also to know the intimate structures of plants and animals; you find that all organisms are composed of living elements—the cells, which in uncountable numbers and inexhaustible variety adapted to the functions of the organs build up the body of all living things. In all structures of the body these cells are glued together in many-fold ways and combinations forming that which we call the tissues. Complicated methods are required to make them accessible for microscopic examination. In one place only, the composing cells of the body present themselves separated to our view—in the blood.

A drop of fresh blood under the coverglass protected from drying by a vaseline border, shows at the first glance in the microscope thousands of elements. Each a cell for itself suspended in the fluid. Studying these particles a little closer you realize that they are not all of the same kind. Predominant are small yellow-red circular discs—the red blood corpuscles; the rest are colorless cells—the white blood corpuscles. A little bit of attention shows that these latter are divided into a smaller and a larger type, each showing difference in architecture. By a more careful gaze on the larger ones you make out that they too can be divided into groups according to the fineness or coarseness of the granules which fill up their bodies. Besides these cells you see numerous small, non-nucleated bodies—the platelets. They disintegrate under your eyes. Using this quick and simple method, you obtain considerable information.

Acquainted with the appearance of normal blood, you soon are able to note changes in pathological conditions, regarding the number, the arrangement, the cellular composition, the shape, activity and dimensions of the cells. There are many instances in which the examination of fresh blood may give you the idea of what to look for by other methods. I have always stressed the examination of fresh blood because abnormalties in shape and size of the blood corpuscles are much more impressive there than in other preparations and because there is certainty that all you see corresponds to the real conditions during life and that no artificial disfiguration leads you astray.

You know that the blood is a complicated and variable thing—not a single fluid. You have frequently heard that the blood is a liquid tissue. You may take this statement as an image but not as a reality. A tissue is a fixed aggregation of cells in a certain order, where the cells live, grow and die and are regenerated in the most marvelous process of multiplication—that of cell division. In normal blood the cells live but neither do they die nor multiply there. The blood cells are liberated elements of the solid blood forming tissues in the bone marrow and in the lymphatic organs, where they form, grow and multiply, then are set free and expelled into the circulating blood. They die outside of the blood, in the spleen, the liver and the different tissues and cavities of the body. In normal blood you find just the terminal stages of a complicated development, the traces of which are sometimes met with in the blood under pathological conditions. Such traces are often of great importance for the physician but the recognition of these findings requires special knowledge and training on the part of the technician. The technique of fixation and of staining are the methods for further study. By the first method we try to protect the cells from disintegration because all living structures are liable to decay and decompose. Using different ways of fixation we are further able to demonstrate hidden structures and make them suitable for stains. Most of the structures in the living cells are resistant to staining and in your routine work you will use stains for living blood cells, known as vital or supravital stains only rarely. Generally you will stain after fixation. Why do we proceed in this way? Fixation is a process of coagulation of the protein substance of the cell and

formation of different chemical compounds of the reagent with different proteins. These compounds will react to the stain each in its own way, because staining is a chemical process.

The dyer in the dyehouse is aware that he cannot use the same dye for wool, cotton and silk. When he wants to do this, he must prepare the yarns accordingly. Now take for instance a mixture of silk, cotton and wool strand, dip it into a tanning solution and afterwards in a dye. The threads will have acquired quite different hues and on first sight you will discern the silk thread, the wool thread and the cotton thread by their respective colors. Similarly you apply a dye consisting of a number of stains to a blood smear—the cells in the blood select the stain appropriate to their chemical constitution and you will recognize each kind of cell and the age of development by the color and degree of absorption of the dyes. Now you will understand why the stained specimen does not only show more distinctly the things already visible in the fresh preparation, but moreover new structures appear, richer and unexpected differences and varieties disclose themselves to you.

A well stained blood-smear gives me a sort of esthetic pleasure, even now after years and years of seeing so many of them, it is a wonderful and a beautiful sight, which you certainly do experience yourselves. Staining is an art and even the routine methods have their moods. To evercome them one requires understanding and the most accurate handling of every step. You should always keep in mind that the slightest lack of exactitude can influence the results. Even the slightest alteration of technique may mislead in the interpretation of the microscopic image. It is a matter of long experience to avoid errors caused by artefacts, but such experience you can only acquire by hard, conscientious work and continued study.

In this way you will soon learn to recognize the cellular elements composing the normal blood in well distinguished types. Occasionally you may encounter a cell, which cannot be readily placed in the accepted system. The reason for it is due to the fact that living structures, and such are the cells of our blood, have a certain range of free play in their morphology. This rule applies still more to pathological conditions and there you will meet with greater difficulties, nevertheless do not become discouraged. Perhaps it will be

a comfort to you to know that even hematologists with the greatest experience are sometimes unable to agree on an exact interpretation. Of course in lectures and in text books you are shown types of cells designated by special names which serve as reliable guides in the wilderness of forms. However such types will never exhaust the variety arising from the fact that all pathologic forms are results of regenerative and degenerative processes, and what you see are just momentary stages of a continuous developmental chain. They are stations in the process, like snapshots of a running horse, which show only single arrested position of the limbs but omit the intermediate ones. Here again understanding is the only help. Knowing the origin of the blood cells and their development in the blood forming organs under normal circumstances, the beginning and the effects of a pathologic degenerative process, you will be able to place the intermediate forms, thereby the snapshots will become intelligible links of a chain of continuous transformation. In this way you may observe the most impressive fundamental features of life, evolution and decay, in the narrow field of the microscope. The increasing reverence for the unfathomable miracles of life and nature, the mounting interest in them will immeasurably enrich your interest in your occupation.

IDENTIFICATION OF DIPHTHERIA ORGANISMS

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Corynebacterium diphtheriae was first described by Klebs in 1882, who studied its morphology from clinical specimens. The organism is commonly known as the Klebs-Loeffler bacillus, accrediting this man who first described it and Loeffler who subsequently cultivated and stained the diphtheria organisms.

The organisms are variable in size and appearance. They are not acid fast, contain no spores, have no flagella or capsules, and are ordinarily Gram positive. The metachromatic granules or volutin which they do form are the main distinguishable feature in stained smears, and the slender rods are often found in V, W, and Z formation due to the characteristic division in reproduction.

Two other organisms closely related; diphtheroids known as Hoffmani and Xerosis, cannot be distinguished morphologically or culturally by the more common methods. These two organisms are never pathological and hence it is incorrect to report a single culture on Loeffler's media as being that of the true diphtheria organisms. To be entirely correct, the report should read "diphtheria-like bacilli present," despite the dissatisfaction which is apt to result from the inadequacy and specificity of such a report.

It is our problem to find some means of determining which organisms are virulent and which are non-virulent in order to give a quick and accurate report for the diagnosis of the disease caused by this organism in which administration of the antitoxin soon after the onset is important in the recovery of the patient. An economical problem lies in the laboratory's two negative reports in order to lift the quarantine; the sooner the laboratory can give these reports the sooner normal living and business conditions can be resumed by those involved.

As early as 1880, diphtheria bacilli have been classified according to their cultural characteristics. Klein described two types of colonies on gelatin. In 1924, Hammerschmidt described two main types. Cowan attributed one of the two types he cultivated in 1927 to be due to roughening and Parker the following year found three types which he also attributed to rough variants. Parish, Whatley, and O'Brien in 1932, classified the diphtheria bacilli on the basis of their ability to ferment starch. Fermentation offers a better index for classification according to Menton, Cooper, Duke, and Fussell, in 1933. Maer, in 1935, classified the organisms according to their ability to ferment starch correlated with their microscopic appearance.

In searching for a substance which would inhibit the growth of contaminating organisms without affecting the diphtheria bacilli, it was found that diphtheria bacilli were very susceptible to the inhibitory action of basic dyes and salts of metalloids. Anderson, et al, in 1931, used small concentrations of potassium tellurite very successfully in classifying three types on media containing this compound, and in 1933, confirmed their earlier work with a study on 500 additional cases. In making up this media the writer used a media similar to that used by Glass. The medium was tested by plating known types from McLeod's medium and found to give sufficiently differential characteristics to be used satisfactorily.

The media is made up by dissolving 6 gms. of Difco Brain-Heart Infusion and 3 gms. agar in 200 c.c. distilled water. 0.5 c.c. of N/1 NaOH is added and the medium sterilized in the autoclave at 15 lbs. for 20 minutes. 2 c.c. of a 2% solution of potassium tellurite is added to each 100 c.c. of medium to give a final concentration of 0.04% potassium tellurite. 20 c.c. of blood is added to the 200 c.c. of medium, cooled to exactly 750 C. to give the desired chocolate blood agar. When the medium has cooled to a lower temperature, plates are poured and kept in the refrigerator, not more than two days. The plates are transferred to the incubator for 20-30 minutes before being planted.

On this media, the diphtheria organisms have been divided into three groups as follows:

Mitis—medium size, domed, grey-black colonies;

Gravis—metallic grey colonies with a peaked center with ridges radiating out to a serrated edge, giving an appearance similar to a "daisy head."

Intermediate—smooth colony with grey-black center and light periphery.

Cocci appear as either (1) black, very rough and wrinkled colonies, (2) smooth black colonies with entire edges, or (3) grey center with light periphera. The cocci can ordinarily be differentiated by the consistency of the growth, as this growth is very sticky and when fished, the colony picks up entirely from the plate; whereas, the diphtheria colony is of granular consistency.

This method of arriving at the true identity of the organism is detailed and requires a series of tests which ordinarily take longer than the time in which the report is most valuable. In searching for a characteristic which would differentiate and identify the true diphtheria organisms from all others in one test, the original study of the production of the enzyme phosphatase by these organisms was undertaken by the writer. The result of this experiment proved this test to be of no value for the purpose.

Without this test which we hope will someday be revealed to us as demonstrating the one characteristic which differentiates the group of true diphtheria organisms from diphtheroids, we can only use the known tests to the fullest extent of their worth and continue to look for this one characteristic which they may not even possess in common. Final reports made on direct smears are apt to be inaccurate unless the patient has reached the severe stage of the disease that the diphtheria organisms have overgrown all the normal flora of the throat and nose. The presence of a few diphtheria-like bacilli in direct smears is normal and yet may be found in patients suffering from the disease, so that a culture in most cases is warranted.

Cultures should be made on both Loeffler's media and Potassium Tellurite Chocolate Blood Agar, which is the best differential media known at the present time. Fresh and moist Loeffler's media should be used and care exercised to avoid the dried tubes. It is important to smear from the actually involved areas, examining the throat with good illumination, and to have the tubes incubated without delay. At the end of 12-18 hours, films are stained with Methylene Blue,

Albert's, or one of the stains of the technologist's preference.

Virulence testing by using two rabbits, one having received antitoxin, and injecting 0.1 c.c. of the culture intracutaneously is the only accurate virulence test that we have. Fermentation tests because of their non-specificity to types are not particularly helpful.

Discussion

Thus we have recognized our problem of finding one characteristic which the true, virulent diphtheria organisms have that is different from the non-virulent diphtheria and diphtheroid organisms. Cultural characteristics on ordinary media give only a division of rough and smooth variants. Fermentation of sugars and starch is not conclusive, and the organisms do not produce phosphatase.

Conclusion

Despite the extensive work done, the most satisfactory—that of using Potassium Tellurite Chocolate Blood Agar for classifying the diphtheria organisms—is not sufficiently specific to warrant its use alone. Until we have discovered the test which will give us the desired information in the shortest time, we shall find our best procedure in the use of the two media described, Loeffler's and Potassium Tellurite, and stained films from these cultures, followed by animal virulence test.

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PATHOLOGICAL PHYSIOLOGY OF THE LIVER*

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The liver is the largest, the most versatile and the most hermit-like organ in the body. Perhaps it is significant, but in what way I do not know, that man has more liver than brains. Because of its size, it has a large factor of safety, or functional reserve. It is possible to remove three-fourths or more of the liver of an experimental animal without serious results to the animal as a whole. Not only so, but its power of regeneration is so great that it will produce new liver tissue so rapidly and so competently that not only is its normal functional level maintained but it soon recovers the greater part of its original functional reserve.

The blood supply of the liver is relatively enormous, and comes from two sources. From one-fourth to one-third is derived from the hepatic artery and is therefore arterial blood; two-thirds to three-fourths reach the liver by way of the portal vein and is venous blood. The blood circulating through this organ therefore contains less oxygen than does the blood of any other organ in the body, and yet, in spite of this continuous moderate anoxemia, it carries on effectively. It is adapted to a low oxygen tension and can perhaps work best under this condition.

The liver has a dozen or more separate and distinct functions. But in spite of its size and its busy activity, it is a most silent and dignified organ. When the heart is called upon to do more work it makes its complaints known by rapid pounding that may cause much distress. When the demand for more oxygen is made upon the lungs, as during vigorous exercise or because of heart disease, they

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* *Given at the Meeting of the Illinois Society of Clinical Laboratory Technicians in connection with the 11th Annual Meeting of the Tri-State Hospital Assembly, Chicago, Illinois.*

comply, but with groans and heaving respiration. The intestines and the kidneys make us acutely conscious of their functional activity. Even the brain produces a thought or calls up a memory once in a while. But the liver, with its great size, its numerous and varied activities, performs its work with quiet and patient industry. Even when insulted by serious injury it only rarely retaliates with severe pain.

The functions of the liver can be grouped into those which maintain the constitution of the blood, and those that are concerned with metabolism.

During fetal life, the liver is an important source of supply of blood cells. It surrenders this function only when the bone marrow has developed sufficiently to take over this important activity. But even then its duties pertaining to the blood are not ended, because during the entire life of the individual it is one of the official cleansers of the circulating blood. Red blood cells are relatively short-lived. As senile erythrocytes totter through the sinusoids of the liver they are quietly removed from the blood stream by the Kupffer cells, to be disposed of by a type of euthanasia. While the liver is not the only organ that performs the last rites for red blood cells dead of old age, it is an important factor in this function. Furthermore, no matter where the red cells are caught up and destroyed by the littoral cells of the widely distributed reticuloendothelial system, the products of that destruction ultimately reach the liver for further disposal. The hemoglobin is decomposed into iron-containing hemosiderin and iron-free hamatoidin, the latter identical chemically with bilirubin. The liver stores much of the hemosiderin, but excretes all the bilirubin as a waste product. It also groups together some of the amino-acids and perhaps other elements which are necessary in the building of new hemoglobin by the bone marrow. In other words, the liver is responsible for the first steps in the manufacture of hemoglobin for new red cells. Lack of anti-anemic factor interferes with this function and pernicious anemia results.

Bilirubin is not manufactured by the liver, but by the cells of the reticuloendothelial system. The liver is merely its portal of exit from the body. When, for any reason, the liver is unable to excrete

this waste product, it accumulates in the blood in sufficient concentration to cause jaundice. Jaundice is of two chief types, obstructive and hemolytic.

In hemolytic jaundice, red blood cells are destroyed in such numbers and with such rapidity that the liver, especially if it has suffered injury, is unable to maintain its excretion of bilirubin at a correspondingly high level and jaundice is an inevitable result. In this type of jaundice the bile pigment present in the blood has not passed through the liver cells. For some reason it usually gives an indirect van den Bergh reaction and it is not excreted in the urine. Hence this type is called acholuric jaundice. On the other hand, when the outflow of bile from the liver is obstructed the secretion is retained in the finer bile ducts and bile capillaries from whence it reaches the lymphatics or passes directly into the blood stream. In any case, the bile pigment that is in the blood plasma in obstructive jaundice has passed through the liver cells. Although the liver cells have nothing to do with the formation of bilirubin, in its passage through them they do work some alteration in it so that it acquires qualities that render it filterable by the kidneys and so is present in the urine and also, it is said, cause it to give the direct van den Bergh reaction. The hemolytic and obstructive types of jaundice differ in another significant respect, namely, in the obstructive type, both bile salts and bile pigment are present in the blood plasma, while in hemolytic jaundice, there is only bile pigment but no bile salts. For this reason the hemolytic type is sometimes called dissociated jaundice.

Bilirubin is carried in the bile to the intestine. It is not normally present as such in the feces for it is reduced by bacteria in the colon to stereobilin which is very similar to, if not identical with, urobilin. A portion of the reduced subtance, urobilinogen, is absorbed from the colon and carried to the liver in the blood of the portal vein. Most of it is re-excreted in the bile but a small portion passes through the liver to the general circulation to be filtered out by the kidneys. Any injury of the liver reduces its power to re-excrete urobilinogen leaving a larger quantity to enter the general circulation. For this reason an excess of urobilinogen in the urine is a sensitive indicator of liver damage.

Of the five factors which are concerned in the coagulation of the blood, three of them are believed to be produced wholly or in part by the liver. These are fibrinogen, heparin or antithrombin, and prothrombin. Heparin is a phospholipid (kephalin) and is produced in the liver but also to some extent in other tissues. This is the substance which prevents calcium and prothrombin from combining to form thrombin. The effects of damage to the liver on the production of heparin are not definitely known because other factors in the coagulation of the blood are either interfered with or their production is reduced by the same conditions.

Prothrombin is apparently produced only by the liver. It is related to vitamin K. Prothrombin is reduced in diseases of the liver, especially in obstructive jaundice in which the absence of bile from the intestine prevents the absorption of vitamin K. Whether this vitamin is prothrombin, or whether it is the substance from which prothrombin is made by the liver cells, or whether its presence is necessary for production of prothrombin is not definitely known. But unless it is present in sufficient amount, prothrombin is not formed in adequate quantity and coagulation of the blood does not take place in the normal time and manner. This is the cause of the frequency of hemorrhage in patients with obstructive jaundice. When vitamin K is administered to jaundiced patients along with sufficient bile salts to insure its absorption from the intestine the prothrombin rapidly increases and the coagulation of the blood becomes normal again.

Fibrinogen is one of the plasma proteins. In its natural state it is in solution in the plasma. In the process of clotting of blood it is precipitated by the action of thrombin in the form of fine meshwork or filaments in which the red blood cells and other formed elements of the blood are entangled. In diseases which are characterized by destruction of liver tissue, as in poisoning by phosphorus or choloroform and in acute yellow atrophy of the liver, the amount of fibrinogen in the plasma is reduced. As a result, when blood of such a patient clots, which it does in the normal time, the clot is soft, does not contract and solidify, and is of little value in the closure of severed blood vessel to stop hemorrhage.

Extensive disease of the liver may therefore affect the coagulation of the blood by reducing the amount of prothrombin or of

fibrinogen or both. If prothrombin is reduced alone, as may be the case in obstructive jaundice, there is not enough of this substance to precipitate all of the available fibrinogen and the clot formed in such blood is soft and ineffective in stopping hemorrhage. In other diseases, both prothrombin and fibrinogen may be reduced with the same result. These are the reasons why hemorrhage is a frequent accompaniment of diseases of the liver, and why the physician orders a determination of the amount of fibrinogen in the blood and of the prothrombin time. There is also a difference in the significance of the results in each case. Reduction in the quantity of fibrinogen is evidence of serious damage to the liver itself. A prolonged prothrombin time may be due either to damage to the liver itself, or to interference with the absorption of vitamin K from the intestine because of the absence of bile in the intestinal contents.

Because the sinusoids and veins of the liver are capable of such enormous distention that they can contain two-thirds of all the blood of the body, this organ is an important factor in regulating the total circulating blood volume. This is important in certain types of shock. Also, since blood leaves the liver at a place about an inch from the heart, the liver serves as a safety reservoir in which blood can be impounded to protect the heart from being overwhelmed with blood which it is unable to keep moving because of inherent handicaps due to cardiac disease.

Furthermore, much fluid escapes from the blood in the liver in the form of lymph, so that the blood which leaves the liver is more concentrated than that which enters it. This serves as a temporary safety device when excessive quantities of water are absorbed from the intestines and when large amounts of fluid are given rapidly by intravenous injection. Most of this water is stored in the tissues, with the necessary quantity of sodium chloride to maintain its isotonicity with the tissue fluids, until it can be excreted by the kidneys. While water is passing into the tissues some of it is squeezed out of the blood into the lymphatics of the liver. But this water then passes by way of the thoracic duct into the left subclavian vein to be returned to the general circulation. Hence in dealing with an excessive intake of water, the liver plays a role in thus temporarily shunting some of the excess out of the blood until it can be better

stored in the tissue spaces awaiting its turn to be finally excreted through the kidneys.

The liver plays several important parts both in the general metabolism of the body as a whole and in the special metabolism of proteins, carbohydrates and fats.

The liver and the muscles, partly because of their mass but chiefly because of their intense chemical activity, produce most of the heat of the body. Removal of the liver from an experimental animal reduces its body temperature to a considerable degree. Furthermore, under normal conditions, because of its great production of heat and because of its complete insulation as a result of its position deep within the body, the internal temperature of the liver is higher than that of any other organ.

One of the specific functions of the liver is the formation of bile acids and their salts. These substances are essential to the digestion of fats, which although they are the chief source of energy, are less readily oxidized than are carbohydrates which are the body's emergency fuel. Hence the liver plays a double role in general metabolism, first and in a direct manner by its part in maintaining the temperature by furnishing a considerable part of the body heat, and, second, indirectly by making possible the absorption of the food element, fat, that supplies the greater part of the energy used by the body.

Proteins taken as food are digested in the stomach and intestines and are broken down into their constitutent amino-acids. These are then absorbed into the capillaries in the mucosa of the intestine and carried thence in the blood to the liver. Here the excess of these amino-acids, over and above the quantity needed for maintaining the tissues of the body, are altered by the liver cells. The non-combustible amino or the NH_2 group is split off from the molecule of the amino-acid to form ammonia leaving a short-chain fatty acid. The ammonia thus produced is combined with carbon dioxide by the liver to form urea which is excreted almost wholly by the kidneys. Extensive injury of the liver, or its experimental removal, results in a rapid reduction of the urea of the blood, if the kidneys are still functioning, and an increase in the amino-acids which are excreted as such in the urine because the liver is unable to deaminize them. Leucine and tyrosine crystals may be present in the urine.

The fatty acid residue of the deaminized amino-acids is transformed by the liver into glucose by the process of gluconeogenesis. Some of the simpler amino-acid residues, such as those of glycine and alanine, are completely converted into glucose. The liver is apparently the only organ in the body that is capable of accomplishing this transformation. Severe injury or destruction of a large portion of the liver substance reduces this function.

A portion of the protein molecule or some of its constituent amino-acids, such as trytophane and tyrosine, may not be absorbed from the intestine but are transformed by the action of bacteria into toxic substances such as phenol, indole and skatole and others. These may be absorbed into the blood stream from the intestinal contents and carried to the liver where they are detoxified and rendered harmless by conjugation in the liver cells with sulphate ions or with glycuronic acid to be excreted by the kidneys, the former as the "etherial sulphates" of the urine. Other poisonous substances either absorbed from the intestines or produced in the body itself are also detoxified in the liver. This organ thus has several functions—detoxification of poisonous substances, serving as a reservoir for excess blood in preventing overburdening of a crippled heart, and aiding in removing excess water from the blood stream—functions which serve to protect the remainder of the body from deleterious effects of various kinds.

The polysaccarrhides of the food are decomposed in the intestine into monosaccarrhides, which are then, like the products of protein digestion, absorbed into the blood and carried to the liver. Here the monosaccarrhides, fructose and gelactose are transformed into glucose. The glucose which was absorbed from the intestine together with that produced by the transformation of fructose and galactose and that formed by the action of the liver upon the deaminized amino-acids from protein digestion are polymerized into glycogen and stored in the liver for future use by the body as a whole. From all these sources the liver acquires, on an ordinary diet, a considerable store of glycogen. The polymerization of glucose is accomplished by the enzyme, glycogenase which is probably a reversible enzyme capable both of building up and of breaking down molecules of glycogen, depending upon conditions. In any case, when sugar is needed by the tissues for energy purposes, the glycogen

is decomposed into glucose which is liberated in the blood stream. There appears to be a condition of homeostasis between the level of blood sugar and the amount of glycogen in the liver, that is, when the blood sugar is at or above the normal level glycogen is not disintegrated, but when the level of blood sugar is lowered glycogenolysis occurs and glucose passes into the blood.

Various conditions can interfere with this balance between liver glycogen and blood sugar. In diabetes mellitus, the absence of insulin interferes with the storage of glycogen in the liver and the blood sugar is constantly at an abnormally high level. In hyperthyroidism, the constant demand for sugar for energy purposes because of the greatly augmented metabolism, causes depletion of the store of liver glycogen, the level of blood sugar may occasionally be higher than normal and some of it may even escape in the urine. The injection of adrenalin or overactivity of the adrenal medulla or of the diabetogenic substance of the pituitary causes a mobilization of glucose and if maintained for any length of time depletes the store of liver glycogen.

The glycogenic function of the liver is closely related to sugar tolerance tests. The normal blood sugar curve in such a test does not rise very high and falls rapidly because the liver is able to transform glucose of the blood into glycogen and thus remove it from the circulation. If the glycogenic power of the liver is lowered, as for instance in the absence of insulin, then the liver is unable to remove sugar from the blood and store it as glycogen at the normal rate, the blood sugar reaches a high level and remains high for a much longer period than normally. The galactose test for liver function is based upon the fact that it is difficult for the liver to transform this sugar into glucose. Since this transformation must be accomplished by the hepatic cells before the sugar can be stored as glycogen, any damage to the liver is likely to interfere with this process. Because of the slower rate at which the transformation is made under such conditions, the galactose remains for a longer time in the blood stream and a greater quantity of it is excreted in the urine.

The liver plays a double role in the utilization of fats by the body. In the first place, it must produce bile salts in order that fat

may be digested and absorbed from the intestine. When bile salts are absent from the intestine as in obstructive jaundice, little or no fat is absorbed. And, unlike the products of digestion of proteins and carbohydrates, which are absorbed into the blood stream and carried directly to the liver, fat enters the lymphatics and only reaches the liver from the general circulation. Having entered the general circulation first, fat is stored not in the liver primarily but in the so-called fat depots, namely the subcutaneous and retroperitoneal tissues and the omentum which are the storage places for fat as the liver is the storehouse of sugar. But there is a further difference, in that the fat is stored in its depots as such, that is, as neutral fat, while sugar is stored in a polymerized form as glycogen. Most of the fat in these depots is saturated fat, because this is the type that predominates in the diet. But saturated fat is rather inert chemically; to be used it must be desaturated. This process is apparently accomplished in the liver. At least, the greater part of that in the liver is unsaturated. It also is probable that fat is transferred from the depots to the liver in appreciable amounts only when the energy needs of the body are not being supplied in the diet. Whether the liver actually is capable of unsaturating fat or whether it has a selective action and takes only unsaturated fat from the blood is not known with certainty. But it is the general opinion, and this is the second role of the liver in the utilization of fat, that the liver does convert inert saturated fat into the chemically active unsaturated form which can be used or burned by the tissues for energy purposes.

There is a sort of reciprocal relationship between fat and glycogen in the liver. In conditions in which the amount of liver glycogen is low, fat may accumulate in large quantities, as in diabetes mellitus. Best and his co-workers have shown that there is a relation between quantity of fat in the hepatic cells and the amount of choline, or substance from which choline may be derived, such as lecithin, in the diet. When choline is deficient, fat accumulates in the liver in large quantities. The presence of an adequate amount of available choline either prevents the accumulation of fat in the liver or causes it to disappear rapidly if it has already been deposited. There is also an interesting relation between the amount of fat or glycogen in the liver and the susceptibility of the liver to certain types of

injury. Chloroform and phosphorus are poisons which have a selective action on the liver. This organ can tolerate much larger quantities of these poisons if it is well stored with glycogen than if its supply of glycogen is replaced by fat.

Diabetic coma is largely the result of ketone substances in the body as a result of a disordered metabolism of fat, which, instead of being burned completely to carbon dioxide and water, is transformed into acetone, oxybutric and diacetic acids. Whether this is apparently the organ in which this process of ketosis takes place. Whether this is the result of disordered function on the part of the liver or is an adaptive mechanism by which this organ attempts to dispose of residues of fat incompletely burned by the tissues, is not fully understood.

What has been said thus far is concerned with the relation of the liver to the metabolism of neutral fats. But it is also concerned with the disposition of other substances that have some of the characteristics of fat, especially cholesterol, which is excreted by the liver in the bile, and is one of the chief constituents of gall stones. The amount of cholesterol excreted is roughly proportional to the quantity of this substance in the blood. This is evident because during pregnancy a hypercholesterolemia usually exists, and approximately 85 per cent of all patients with gall stones are women and about the same proportion of women who have gall stones have borne children. It is doubtful whether either hypo- or hyper-cholesterolemia is a measure of liver function. However, it is possible that diseases of the liver may interfere with the excretion of this lipoid or at least alter the proportion of cholesterol and cholesterol esters in the blood.

The liver is the most versatile organ in the body. It produces blood cells during fetal life; it aids in clearing the blood of wornout red blood cells, stores the iron obtained from them and excretes the waste product bilirubin; it is an important factor in the regulation of the blood volume and serves as a protective reservoir against overburdening the heart; it is the place of storage of vitamin A, D and K and of the anti-anemic factor essential to the maturation of red blood cells; it is the source of much of the body heat; it secretes bile acids which are necessary for the absorption of fats; it deaminizes amino-acids derived from the digestion of proteins, and forms

urea out of the ammonia thus released; it transforms the short-chain fatty acid residues of the deaminized amino-acids into sugar by the process of gluconeogenesis; it detoxifies many poisonous substances, especially those produced by bacterial action upon the products of protein digestion in the intestines; it produces ketone bodies when fat metabolism is disturbed, as in diabetes; it stores carbohydrates as glycogen and regulates the level of blood sugar by glycogenolytic activity; it excretes cholesterol. These functions are well established. In addition, there is some evidence that the liver is the source of fibrinogen, heparin and prothrombin, elements that are concerned in the coagulation of the blood. It may also desaturate the saturated fats taken in with the food to render easy their utilization by the cells of the body. It becomes clogged with fat when the supply of choline is inadequate.

The location of these numerous functions within the liver lobules is unknown. There is nothing in the microscopic appearance of the liver cells in different parts of the lobules to indicate that they perform different functions. Little has been learned concerning the location of these various functions within the hepatic lobules from the more or less selective action of different poisons upon the liver cells, for the necrosis which accompanies poisoning by chloroform and phosphorus and the changes seen in eclampsia and acute yellow atrophy, while they affect in a measure different parts of the lobules, all have much the same effect on the reduction of the functional capacity of the liver.

The liver is also a very patient organ. It may suffer severe and extensive damage without making detectable complaint either in the form of pain or evident disturbance of function. Because of its great functional reserve, it continues to work with a very considerable degree of efficiency in spite of extensive damage and loss of substance, and refuses to reveal its sufferings clearly by even the most elaborate tests. Because of its multiplicity of functions, there is an equal or greater multiplicity of tests designed to determine its functional capacity. Because there are no criteria by which we can determine which cells of the liver deaminize amino-acids, which detoxify poisons or which produce ketone bodies, may it not be possible that this huge and patient organ has not learned the prin-

ciple of division of labor and that its cells are like the old-fashioned housewife who did all the cooking, serving and dishwashing? If this be true, then it would appear that any good test of liver function, such as the galactose test which determines the liver's ability to transform this sugar into glucose and store it as glycogen, or the test for urobilinogen in the urine which reveals the ability of the liver to re-excrete this substance, is all that is needed to learn the functional capacity of the liver. For when one function of the cells is disturbed or decreased it is likely that all of the other activities of the cells suffer in a similar manner.

ABSTRACTS

LEUCOCYTE COUNT AND RECOVERY FROM TUBERCULOSIS: C. H. Boissevain & E. N. Chapman, Am. Rev. Tuber., Vol. 44, No. 1, July, '41, p. 58.

Studies made at altitudes of 1,000 ft. and 6,000 ft. showed high correlation between the outcome of the disease and the total leucocytes or the Medlar index. In the work done at 1,000 ft. a correlation between the number of monocytes and the outcome was also observed. Patients with a neutrophile count over 6,000 showed a lower death rate at an altitude of 6,000 than at 1,000 ft.

PROGRESS IN THE STANDARDIZATION OF STAINS. NO FURTHER CERTIFICATION OF GENTIAN VIOLET: H. J. Conn, Stain Tech., Vol. 16, No. 4, Oct., '41, p. 141.

After January 1, 1942, the Stain Commission for Certification will no longer certify preparations labelled "gentian violet" as the term is too indefinite. They must be labelled "crystal violet," "methyl violet 2B", etc. As a help in ordering, the last edition of "Biological Stains" suggested "Users should specify crystal violet for bacteriological work and for histological work where a deep blue-violet is required; but should order methyl violet 2B in histological procedures where a reddish shade is called for."

PERMANENT STAINED PREPARATIONS OF THICK BLOOD FILMS: W. Gingrich, Stain Tech., Vol. 16, No. 4, Oct., '41, p. 159.

Films are stained and laked in dilute Giemsa, rinsed in water and dried. May-Gruenwald's stain is applied and slides are again rinsed and dried. MacNeal's tetrachrome stain in methyl alcohol and glycerine may replace Giemsa and a solution of methyl alcohol may be used for May-Gruenwald stain. Mounted in Diaphane films treated this way may have shown no fading in 3 years.

THERAPEUTIC PROBLEMS IN WATER BALANCE FROM THE VIEWPOINT OF THE SURGEON: U. Maes and H. A. Davis, New Orleans Med. & Surg. Jr., vol. 94, No. 5, Nov., '41, p. 207.

A discussion of the various types of dehydration and edema with suggestions for their alleviation is given. Laboratory findings in dehydration are hemoconcentration, increase in the blood N.P.N. and sugar and increase in the CO_2 content. Sodium and chloride values may be extremely low if there is electrolyte loss. If water alone is given in electrolyte loss, cramps in the muscles, muscle fatigue and nausea and vomiting may occur. When dehydration is also due to loss of electrolytes, administration of isotonic solutions may result in diuresis because the solution is hypertonic to the body fluids. Administration of desoxycorticosterone acetate in addition will cause the retention of sodium chloride in the tissues and enable them to increase their water content again. Vitamin B_1 is excreted in the urine and administration of intravenous fluids will accelerate its loss, leading to loss of appetite with numbness and tingling sensations in the extremities.

METABOLIC STUDIES IN PATIENTS WITH CANCER OF THE GASTRO-INTESTINAL TRACT. I. PLASMA VITAMIN A LEVELS IN PATIENTS WITH MALIGNANT NEOPLASTIC DISEASE, PARTICULARLY OF THE GASTRO-INTESTINAL TRACT: J. C. Abes, A. T. Gorham, G. T. Pack and C. P. Rhoads, Jr. Clin. Inves., vol. XX, No. 6, Nov., '41, p. 749.

Levels below normal were found in 86% of the cancer cases. Observations showed that inadequate ingestion or malabsorption would not account for these low levels. A possible explanation is a hepatic dysfunction in the storage of vitamin A or its formation from carotene. Patients who had had successful resection of gastro-intestinal cancer showed low levels much less frequently. Low levels were also found in patients with lymphomas, cancer of the head of the pancreas, and bone sarcoma.

AN IMPROVED DILUTION FLUID FOR ERYTHROCYTE COUNTS: L. Vallarin, Stain Tech., Vol. 16, No. 4, Oct., '41, p. 177.

This solution stains erythrocytes brown and destroys leucocytes. Formula: iodine 0.3%; potassium iodide 0.4%; sodium citrate ($Na_3C_6H_5O_7$ $2H_2O$) 1%. Dissolve KI in 50 ml. distilled water. Add iodine crystals using gentle heat. Add citrate, make up to 100 ml. and filter.

The iodine stain was easily removed from glassware. The solution was not appreciably affected by 10 days' exposure to direct sunlight.

A SIMPLE AND ACCURATE METHOD FOR THE DETERMINATION OF CHLORIDE IN BIOLOGICAL FLUIDS: O. Schales and S. Schales, Jr. Biol. Chem., Vol. 140, No. 3, Sept., '41, p. 879.

The chloride-containing material is titrated with standard mercuric nitrate solution using diphenylcarbazone as the indicator. The end-point is purple and is sharp and easily read. Protein need not be removed but removal does intensify the end-point.

LABORATORY PROCEDURES IN INTESTINAL TUBERCULOSIS: A. L. Kruger & H. J. Perlberg, Am. Rev. Tuber., Vol. 44, No. 1, July, '41, p. 73.

Woldman's phenolphthalein test is discussed. The results of this investigation do not substantiate it.

Stool examinations were found of value only in ruling out amoebiasis and other similar conditions.

The diagnosis on intestinal tuberculosis may be based on positive sputum findings, or a history of them and comparison of symptoms with those known to occur in intestinal tuberculosis and by the X-ray observation of the ileocaecal spastic filling defect characteristic of tuberculosis.

A SYNTHETIC MEDIUM FOR THE CULTIVATION OF STREPTOCOCCUS FECALIS: R. L. Schuman & M. A. Farrell, Jr. Inf. Dis., Vol. 69, No. 1, July, '41, p. 81.

Str. fecalis was found to grow well on a completely synthetic

medium consisting of pantothenic acid, vitamin B_6, riboflavin, glucose, a salt mixture, arginine, glumatic acid, methionine, tryptophane, tyrosine and valine.

TRAY FOR STAINING TUBERCLE BACILLI: W. Steenken, Jr., Am. Rev. Tuber., Vol. 44, No. 1, July, '41, p. 115.

A tray of glazed porcelain in which each slide is placed in a separate compartment is described. This would eliminate contamination of negative slides. The amount of stain used is about that required to flood. The entire unit may be heated in the oven. Trays are cleaned with sulfuric acid cleaning solution.

PRESENT STATUS OF THE TUBERCULIN PATCH TEST: C. Keresztur, Am. Rev. Tuber., Vol. 44, No. 1, July, '41, p. 94.

The literature concerning this test is reviewed with the conclusion that at present it is not sufficiently reliable to replace the Mantoux test.

THE PHYSIOLOGY OF CAPSULATED STREPTOCOCCI: J. E. Morison, Jr. Path. & Bact., Vol. LIII, No. 1, July, '41, p. 1.

Physiological variations of pH and concentrated salt solutions showed little effect on capsules. Heat destroyed them rapidly, presumably by enzyme autolysis. This enzyme-like substance was separated and its activity demonstrated. Formalin was found to influence its capsule destruction somewhat.

"PRIMARY" HYPOCHROMIC ANEMIA TERMINATING IN PERNICIOUS ANEMIA: E. B. Miller, W. Dameshek, Arch. Int. Med., vol. 68, No. 3, Sept., '41, p. 375.

Reference is made to a previous article reporting either complete achlorhydria or distinct hypoacidity in most cases of "primary" or "idiopathic" hypochromic anemia. A case is presented of the hypochromic type which responded well to iron therapy but developed a typical pernicious anemia with response to liver therapy several years later. In the second case the "hypochromia" picture was not com-

pletely typical. The mean corpuscular volume was normal, there were some macrocytes and the bone marrow picture was atypical. Administration of iron removed the low color index and finally brought about hyperchromia and macrocytosis. The explanation given is that deficiencies of both iron and liver extract were present simultaneously.

CLINICAL LIPOID NEPHROSIS: G. G. Gilbert, Arch. Int. Med., vol. 68, No. 3, Sept., '41, p. 591.

Detailed report of a case of lipoid nephrosis in a child of 5 years with laboratory findings following the various medications tried. N.P.N. varied from 15-342 mg.%, total protein from 2.61-500 mg. per 100 cc., cholesterol from 182-1,140 mg. per 100 cc. Throughout the observations urinalyses gave 3-4 plus albumin reactions and were positive for WBC, RBC and casts though they varied somewhat.

The terminal picture, together with the autopsy findings was that of typical glomerulonephritis, substantiating the theory that lipoid nephrosis is merely a stage in the development of glomerulonephritis and not a separate entity.

A CASE OF URINARY MYIASIS: A. C. Candler, Jr. Parasitology, vol. 27, No. 5, Oct., '41, p. 465.

A note of the recovery of third-stage larvae of a Lucilia from a urine sample. Other findings were many leucocytes and erythrocytes, 4+ albumin and a heavy mixed flora of motile bacilli.

EXTRATHYROIDAL IODINE METABOLISM: A. Chapman, Endocrinology, vol. 29, No. 5, Nov., '41, p. 686.

Intact and thyroidectomized rats were maintained on diets low in iodine and adequate in iodine with the result that in the intact animals the iodine intake had little effect while in the thyroidectomized group the difference in iodine level altered the changes in weight and surface area, water intake, food utilization and the metabolic rate. The author concludes that iodine may be important in the absence of thyroid tissue for the production of thyroxine-like substance in the tissues.

DEMONSTRATION BY THE ELECTRON MICROSCOPE OF THE COMBINATION OF ANTIBODIES WITH FLAGELLAR AND SOMATIC ANTIGENS: S. Mudd and T. F. Anderson, Jr. Immun., vol. 42, No. 3, Nov., '41, p. 251.

The authors present a group of photographs of electron microscope films of E. typhosa, S. paratyphi A and B and B. subtilis treated with homologous and heterologous sera. With the specific antiserum, the flagella become thicker, less sharp and less uniform in outline and tend to cohere because of the deposition of antibodies upon them. Cell walls become more opaque and fuzzy in outline. The average thickness of flagella of E. typhosa was 0.72 mm. at a magnification of 25,000 times, of B. subtilis, 0.465 mm.

THE GROWTH OF COLIFORM BACILLI IN DISTILLED WATER: J. W. Bigger and J. H. Nelson, Jr. Path. & Bact., vol. LIII, No. 2, Sept., '41, p. 189.

Natural waters which did not support growth of coliform bacilli could be made growth-supporting by filtration. Contact with rubber was not necessary to do this. Distilled water may be made to support growth by contact with rubber tubing due to the talc on the tubing, though the talc itself does not supply the nutritive materials. As growth does not occur in vacuo or in oxygen, hydrogen or nitrogen, the nutritive materials are apparently derived from the atmosphere and the suggestion is made that they may be carbon dioxide and ammonia. Out of 75 inorganic substances tested, 20 had the same effect as talc.

THE ADHESIVENESS OF BLOOD PLATELETS IN NORMAL SUBJECTS WITH VARYING CONCENTRATIONS OF ANTICOAGULANTS: H. P. Wright, Jr. Path. & Bact., vol. LIII, No. 2, Sept., '41, p. 255.

A method of measuring the adhesiveness of platelets is given. The effect of various concentrations of heparin, sodium oxalate and two chlorazol dyes was studied with the observation that the greater the concentration of these substances, the less the adhesiveness of the platelets. The fall in platelet counts of blood in vitro was shown to be due chiefly to their sticking to the glass walls of the container.

BOOK REVIEWS

DISEASES OF THE BLOOD AND ATLAS OF HEMATOLOGY
With Clinical and Hematologic Descriptions of the Blood Diseases Including a Section on Technic and Terminology, by Roy R. Kracke, M.D., Professor of Bacteriology, Pathology and Laboratory Diagnosis, Emory University School of Medicine, Pathologist to the Emory University Hospital. Consultant in Hematology to the Grady Hospital and Eggleston Hospital for Children, Atlanta, Ga. Formerly Director of the Hematological Registry, American Society of Clinical Pathologists. Second edition, thoroughly revised, reset and enlarger, 692 pages, including 54 color plates and 46 other illustrations. Price $15.00. J. B. Lippincott Company, 1941, Philadelphia, London, Montreal.

With the increased interest in and application of hematology to clinical practice it is evident that a need for a complete reference work on the subject was paramount. This authoritative work by Kracke has met the need most admirably. Many new developments in hematology in the past four years has necessitated a thorough revision in the second edition. New material has been added on fractionation of liver extract, the action of drugs on the blood, hemoglobin and its derivatives and on the porphyrin compounds; there are new chapters on the hemolytic anemias, on hemoglobinuria and hemoglobin. With the advent of war and with the large increase in the number of blood transfusions being given a timely chapter on blood groups and blood and plasma transfusions and the operation of a blood bank has been added. Several methods for the dessication of plasma or serum are mentioned but, as in other chapters, an extensive authoritative bibliography directs the reader to the complete information desired. A chapter on vitamin K gives an account of its discovery, distribution in nature, physiology of action, conditions causing deficiency and other recently discovered facts. Certain omissions of the first edition have now been corrected including

material on osteosclerotic anemia, achrestic anemia, ovalocytosis, Hodgkins disease and histoplasmosis. A new chapter includes recent advances in the use of radiation and radioactive isotopes in the treatment of leukemia. Ten new color plates and 29 other illustrations have been added. Inasmuch as Wright's stain is the most universally used in the United States colored plates are so shown except where other staining is indicated. The eight sections with their 46 chapters and numerous subheadings include hematologic terminology, the development and morphology of blood cells, leucocytosis and leucopenia, the anemias, the leukemias, hemorrhagic diseases, hematologic technic and an important section on miscellaneous conditions including the chapters on transfusions, Hodgkin's disease and the blood picture of normal laboratory animals.

PRACTICAL METHODS IN BIOCHEMISTRY, by Frederick C. Koch, Frank P. Hixon, Distinguished Service Professor of Biochemistry, University of Chicago. Third edition (revised), pp. 314. The Williams and Wilkins Company, Baltimore, Md., 1941. Cloth, price $2.25.

Devised for medical students this laboratory manual presents the more important qualitative and quantitative aspects of cell constituents, of cell activities and of the composition of blood, secretions, and excretions. In this third edition new methods on uric, lactic and pyruvic acids have been added. Part I on the chemistry of cell constituents includes experiments on carbohydrates, lipins, proteins, hydrogen ion concentration, nucleoproteins and nucleic acids. Part II takes up experiments on the chemistry of salivary, gastric and intestinal digestion and bile and Part III includes the quantitative analysis of blood and urine. An appendix of general laboratory instructions gives the preparation of all reagents and solutions used for experiments in the manual. There are 232 experiments concisely given.

NEWS AND ANNOUNCEMENTS

TO MEMBERS OF THE AMERICAN SOCIETY OF MEDICAL TECHNOLOGISTS

The Program Committee is searching for talent among the membership—for those who might contribute suitable scientific papers at the 1942 Convention.

In order that this program may be truly representative of the membership, we urge your cooperation in bringing to the attention of this Committee, names of members engaged in various branches of medical technology.

The American Society of Clinical Pathologists has offered an award of fifty dollars to the technologist for the best scientific paper presented at the 1942 Convention to be held in Philadelphia, Pa., on June 8, 9, and 10. Only members of the American Society of Medical Technology are eligible for the award.

Each suggestion will be carefully considered by the Committee and choices made in an effort to give you the very best program possible.

Please send in names, suggestions, and any information you deem worthy of consideration.

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TRANSLATED AND REPRINTED FROM "FINLAY," OFFICIAL PUBLICATION NAT. ASSO. LAB. TECH. OF THE REPUBLIC OF CUBA, VOL. 1, NO. 9, OCTOBER, 1941

"Carlos J. Finlay" Prize: The Chief of Education and the Minister of Education have created a prize which shall be called "Carlos J. Finlay," consisting of a trip to the United States for two months' study for laboratory technicians who have accomplished the most during the course. The judges will include the Head of Rural Education, the Head of the Central Laboratory and the Director of the Control Department of the Clinical Laboratory of the Bureau of Rural Education, who will report to the Minister of Education their decision regarding the awarding of the prize, which will be carried out through the mediation of the American Society of Medical Technologists.

MISSISSIPPI VALLEY MEDICAL SOCIETY 1942 ESSAY CONTEST

The Mississippi Valley Medical Society offers annually a cash prize of $100.00, a gold medal, and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics) and practical value to the general practitioner of medicine. Certificates of merit may also be granted to the physicians whose essays are rated second and third best. Contestants must be members of the American Medical Association who are residents of the United States. The winner will be invited to present his contribution before the next annual meeting of the Mississippi Valley Medical Society at Quincy, Ill., Sept. 30, Oct. 1, 2, 1942, the Society reserving the exclusive right to first publish the essay in its official publication—the *Mississippi Valley Medical Jour-*

nal (incorporating the *Radiologic Review*). All contributions shall not exceed 5000 words, be typewritten in English in manuscript form, submitted in five copies and must be received not later than May 1, 1942. The winning essay of the 1941 contest appears in the January, 1942, issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.). Further details may be secured from Harold Swanberg, M.D., Secretary, Mississippi Valley Medical Society, 209-224 W. C. U. Building, Quincy, Illinois.

FELLOWSHIPS FOR RESEARCH IN NUTRITION

Scientific attack on problems of the American diet was furthered today with the announcement by Charles H. Swift, chairman of the board of directors of Swift & Company, of the establishment of a series of fellowships for research in nutrition. The fellowships are intended to aid the federal government in its long-range national nutrition program.

The fellowships provide for special research to be undertaken in laboratories of universities and medical schools with funds which the company has set aside as grants in aid, beginning November 1. The fellowships will be for one year but may be renewed where the project warrants it.

Any fundamental study of the nutritive properties of foods or the application of such information to improvement of the American diet and health will be eligible for consideration for a grant, according to Dr. R. C. Newton, vice-president in charge of the company's research laboratories, who will coordinate the program.

"A higher level of nutrition for the better health of all Americans is an integral part of national progress," Mr. Swift pointed out in makng the formal announcement. "To advance fundamental knowledge of foods and to discover or develop ways to feed our nation better and make our people healthier, happier, and more efficient, Swift & Company has undertaken to expand its support of nutrition research. The fellowships in nutrition are designed further to enlist the country's research talents and facilities in order to achieve the long-range objectives of the national nutrition program and the immediate aims of national defense."

Illinois

Officers of Illinois Society of Clinical Laboratory Technicians for 1941-1942:

President—Mr. J. H. Richardson, 3510 W. 61st Place, Chicago, Illinois.

Vice-President—Miss Lucille Moore, Carle Hospital Clinic, Urbana, Illinois.

Treasurer—Miss Margaret Mellody, Passavant Hospital, 303 E. Superiir St., Chicago, Illinois.

Secretary—Miss Edna H. Murmann, 3934 N. Monticello Ave., Chicago, Illinois.

Minnesota

CONTINUATION COURSE

The Continuation Course in Medical Technology held at the University of Minnesota's Center for Continuation Study in October, 1941, was attended by more than 80 technologists, half of whom were from outside the state.

In addition to the many lectures, very excellent demonstrations in bacteriology and chemistry were presented. Photelometric methods were stressed in chemistry but many of the methods given can be adapted to the colorimeter.

Dr. Frank Heck, of Rochester, speaking on "Abnormal Blood Morphology" rather surprisingly devoted a portion of his illustrated lecture to the proper making and staining of blood smears. He stressed the point that a poor preparation makes a morphological study impossible.

Another unusually interesting lecture was the one given by Dr. Arthur Henrici on the "Diagnosis of Fungus Infections." This was accompanied by lantern slides showing clinical and laboratory features of the conditions considered. Many of the pictures were of slide preparations mounted in Amman's fluid, which Dr. Henrici is to a large extent substituting for strong hydroxide solutions.

Mimeographed note books, with detailed directions for the methods demonstrated and containing some of the shorter lectures in toto were prepared for the enrollees and made extensive note-taking unnecessary.

Many of the technologists in the state will be glad to know that the Continuation Course may be made an annual affair.—From the *Minnesota Medical Technologist.*

The Minnesota Medical Technologist has entered upon its fourth year of publicity and for the Christmas number came for the first time in a white cover trimmed with green scrolls and print. The state no doubt takes pride in the editor, Miss Mary Conroy, and associate editor, Mrs. Martha Strolberg's fine work. The paper carries two solid pages in advertising which advertising has sustained the financial burden of the publication for a large part.

The Standing Committee chairmen for the Minnesota Society of Medical Technologists for the present year are: Constitution—Mrs. Martha Strolberg; Standard and Studies—Miss Catherine Hanitch; Membership—Mrs. Margaret Keogh; Nominating—Miss Vivian Larson; Convention Program—Miss Agnes Hilden.

Nebraska

The annual fall meeting of the Nebraska Society of Medical Technologists was held Friday and Saturday, December 5th and 6th, 1941, at the University of Nebraska, College of Medicine, Omaha, Nebraska.

Program: December 5, 1941

Registration

34 medical technologists from throughout the state were present.

"Biochemical Determinations of the Inorganics," Roundtable discussion, by Dr. Violet Wilder, University of Nebraska, College of Medicine, Omaha.

"Biochemical Determinations of the Vitamins," by Dr. Sergius Morgulis, Professor of Biochemistry, University of Nebraska, College of Medicine, Omaha.

"Normal Blood," by Dr. John S. Latta, Professor of Anatomy, University of Nebraska, College of Medicine, Omaha.

"Leukemias," by Dr. G. W. Covey, Lincoln, Nebr.

Program: December 6, 1941

Registration

38 medical technologists were present.

"Streamlining Nutrition for You," by Dr. Ruth Leverton, Dept. of Home Economics, University of Nebraska, College of Agriculture, Lincoln.

"Laboratory Evidence of the Effect of Atmospheric Changes on Hospital Patients," by Dr. J. D. LeMar, Assistast Professor of Public Health University of Nebraska, College of Medicine, assisted by Arnold Myrabo.

"Report of the University of Minnesota Continuation Study Course," by Harriet Paige, M.T., Clarkson Hospital, Omaha.

"Public Health Importance of Leptospirosis," by Dr. O. F. Reihart, Omaha.

"America's First Contribution to Civilization," by Dr. C. M. Wilhelmj, Dean of College of Medicine, Creighton University, Omaha.

Pennsylvania

The Ninth Annual Banquet of the Pennsylvania Society of Medical Technologists and Laboratory Technicians was held on Monday, November 10th, at Whitman's in Philadelphia. A goodly number were present; among them we had two guests from the Pittsburgh branch.

The president, Kathleen Cornell, introduced the toastmistress, Henrietta Lyle, who carried on in a very capable manner.

Dr. John A. Kolmer of Temple University School of Medicine was the guest speaker; his talk dealt with the value of the Medical Technologist to the Clinician.

The dinner also provided us with the occasion for the unveiling of our newly acquired charter.

The Benjamin Franklin
PHILADELPHIA
(Headquarters)

Tenth Annual Convention American Society of Medical Technologists

JUNE 8, 9, 10, 1942